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REVISION SERIES

THE THEORY AND PRACTICE OF ELECTRODIAGNOSIS

(*concluded*)

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THE use of electromyography in addition to the estimation of nerve conduction and the plotting of intensity–duration curves often only confirms the diagnosis arrived at by these simple methods. However, electromyographic techniques are unrivalled in the detection of early lower motor neurone regeneration and combined lower motor neurone and muscle-fibre lesions, and in the diagnosis of the rarer neurological syndromes such as the myotonias and myasthenia gravis. Further, the detection of isolated motor-unit potentials may supply the only evidence of continuity of an injured nerve.

CLINICAL ELECTROMYOGRAPHY

As with other methods of electrodiagnosis, a careful technique is essential. In the case of electromyography this consists substantially in sampling a weak or wasted muscle in an endeavour to detect and identify various action potentials, the absence of which is rarely significant but the presence of which can supply the diagnosis.

THE ELECTROMYOGRAPH

Many systems of detection, amplification, and recording of muscle-action potentials have been described, but for clinical work standard equipment is now available commercially. This consists of a needle electrode, single-channel amplifier, cathode-ray tube, and loud-speaker. Such equipment is quite adequate for routine clinical electrodiagnosis, and in this article, therefore, only brief reference will be made to more elaborate recording systems.

ELECTRODES

Since Adrian and Bronk (1929) introduced the single-core needle electrode (concentric or coaxial) and Weddell, Feinstein, and Pattle (1944) showed the value of their directional properties, these needles have become the most widely used. It has been shown that muscle potentials derived from coaxial needles are of comparable duration with those derived from monopolar electrodes but of lower amplitude (Christian, 1954). In contrast, potentials derived from double-core needle electrodes show a marked reduction in duration (Petersen and Kugelberg, 1949). The use of double-core needle electrodes is consequently largely confined to the study of single muscle-fibre potentials or where it is necessary to minimize distant motor-unit interference.

Surface (skin) electrodes are not suitable for qualitative clinical electromyography. They produce an integration of the detected potentials and do not, therefore, allow accurate study of their form. Their use is therefore confined to quantitative studies of muscle action, notably in establishing the linear relationship of the integrated electromyogram to the tension of muscle contracting voluntarily and isometrically (Lippold, 1952) and for measurements of muscle contraction in response to supramaximal percutaneous nerve stimulation (Hodes, Larrabbee, and German, 1948).

AMPLIFIER

The need for accurate amplification is obvious, and equipment avoiding excessive cutting-off of the high or low frequencies must be avoided (Buller and Styles, 1952). In general, it may be said that a differential amplifier (push–pull circuit) with a linear response of from 40 to 4,000 cycles per second (c.p.s.) should be used, and such an amplifier should have a high discrimination ratio against in-phase signals (e.g. 1,000–1) and a low noise level. It should allow potentials in the amplitude range 10 microvolts (μV) to 5 millivolts (mV) to be recorded. Transistor amplifiers are now coming into use (George, 1956). In the absence of differential amplifiers, double-channel systems may give rise to apparent synchronization of motor-unit activity (Lundervold, 1949).

RECORDING SYSTEMS

In general, a cathode-ray tube of 6 inches or more diameter and a loudspeaker are all that is necessary. While the majority of muscle-action potentials are readily identified by their sound, a visual check is necessary, particularly if confusion between long-duration and short-duration polyphasic motor-unit activity is to be avoided. Routine photographic recording of detected potentials has little to commend it, but, if required, continuous film or paper recording is the most satisfactory. Isolated film exposures synchronized with the time-base

of the cathode-ray tube are rarely satisfactory because of the difficulty of exposing for transient phenomena. For the more accurate study of muscle-action potentials in clinical work two technical advances are now available. In one, potentials are recorded on wire (Dunn, Bennett, and McIntyre, 1948) or on magnetic tape and studied on replay. Bauwens (1950) has designed an ingenious system in which a magnetic tape loop locked into a triggered time-base allows repetitive replay of potentials for visual study and, if necessary, photographic recording. It is the identification of polyphasic and short-duration motor-unit potentials which presents most difficulty, and because of this Richardson (1951a) suggested the use of frequency analysis. His system was based on the use of electronic filters which filtered off the frequency components of muscle potentials above 400 cycles and measured them. An increase in this measurement compared with that of the low frequency indicated short duration and/or polyphasicity of the detected potentials. Walton and Osselton (1952) have given an account of the use of an audiofrequency spectrometer in which 27 frequencies between 40 and 10,000 cycles were measured. Equipment now commercially available matches the potentials recorded on one beam of a double-beam cathode-ray tube with a histogram on the second beam showing the following frequency components quantitatively: 0·1, 0·15, 0·225, 0·337, 0·505, 0·757, 1·14, 1·7, 2·55, 3·83, 5·75, and 8·6 kilocycles.

It is obvious that the more complicated the equipment the more likely are technical errors, and in particular it is necessary to avoid interference by 50-cycle mains and diathermy. The only other common interfering potential is that derived from the cardiac impulse, but this rarely creates difficulty except with the use of surface electrodes near the chest.

Classification of Muscle-action Potentials

From consideration of the normal electromyographic pattern and the various potentials which are obtained in pathological conditions, neuromuscular potentials may be classified as spontaneous potentials, potentials produced on minimal volition, and potentials produced on maximum volition —that is, the recruitment of the motor-unit potentials.

Electromyography of Normal Muscle

Normal muscle at rest is electrically silent when examined by clinical electromyographic techniques. Voluntary contraction is accompanied by motor-unit discharges of diphasic and triphasic (80–90%) or polyphasic potentials whose average duration in limb muscles (standardized on biceps brachii) is in the range of 5 to 10 msec., whose amplitude is about 1–2 mV

(Petersen and Kugelberg, 1949) (Fig. 6*a*), and whose frequency components lie predominantly in the 0·1–0·7 kilocycle per second range. Facial muscles exhibit potentials whose mean duration is 40–45% less than that of the biceps brachii (Buchthal and Rosenfalck, 1955). Durations increase with age, almost doubling from age 2 to 70 (Buchthal and Pinelli, 1951). The various forms of motor-unit potentials are due to slight staggering of the contractions of the muscle fibres, probably produced by a combination of differences in the length of the branches of the lower motor neurone combined with differences in end-plate transmission, and differences in the distance from the recording electrode of the muscle fibres constituting the motor unit and their end-plates. As the strength of the contraction is increased, the motor-unit potentials initiating voluntary contraction are joined by other similar motor-unit potentials discharging at a rate of up to 20–50 a second. At maximum contraction the tetanic asynchronous discharge of the motor units within range of the needle electrode summates to form the normal interference pattern. This is characterized by a low rumbling sound if demonstrated audibly, or a completely disturbed base line if demonstrated on an oscillograph (Fig. 7*a*). On fatigue it is the large motor units that survive the smaller ones.

SPONTANEOUS MUSCLE-ACTION POTENTIALS

Spontaneous muscle-action potentials are, of course, obtained in the absence of volitional effort by the patient, and, while many are stimulated by movements of the needle electrode in the muscle, the insertion activity which immediately follows movements of the needle is difficult to interpret although occasionally useful in diagnosis (Kugelberg and Petersen, 1949).

There are, strictly, six types of spontaneous potentials to be found in muscle, but also to be considered in this section are tremors and the pseudo-spontaneous activity of cross-reinnervation.

Fibrillation Potentials

These spontaneous action potentials have a duration of 1 to 1·5 msec. and an amplitude of 20–300 μV (usually less than 100 μV) and appear mostly as diphasic spikes (Fig. 5*a*). Their frequency is in the 2–30 per second range, and they are most readily recognized by the regular clicking sound they produce. A further useful characteristic is that in time their frequency tends to drop before they cease altogether. They occur in lower motor neurone degeneration and are due to contraction of individual muscle fibres in response to mechanical (e.g. needle electrode) or chemical (e.g. acetylcholine) stimuli. They are unaffected by cholinesterase (Mead, 1947) or curare (Landau, 1951), and are most easily detected in a warm limb, particularly one in which the denervated muscle

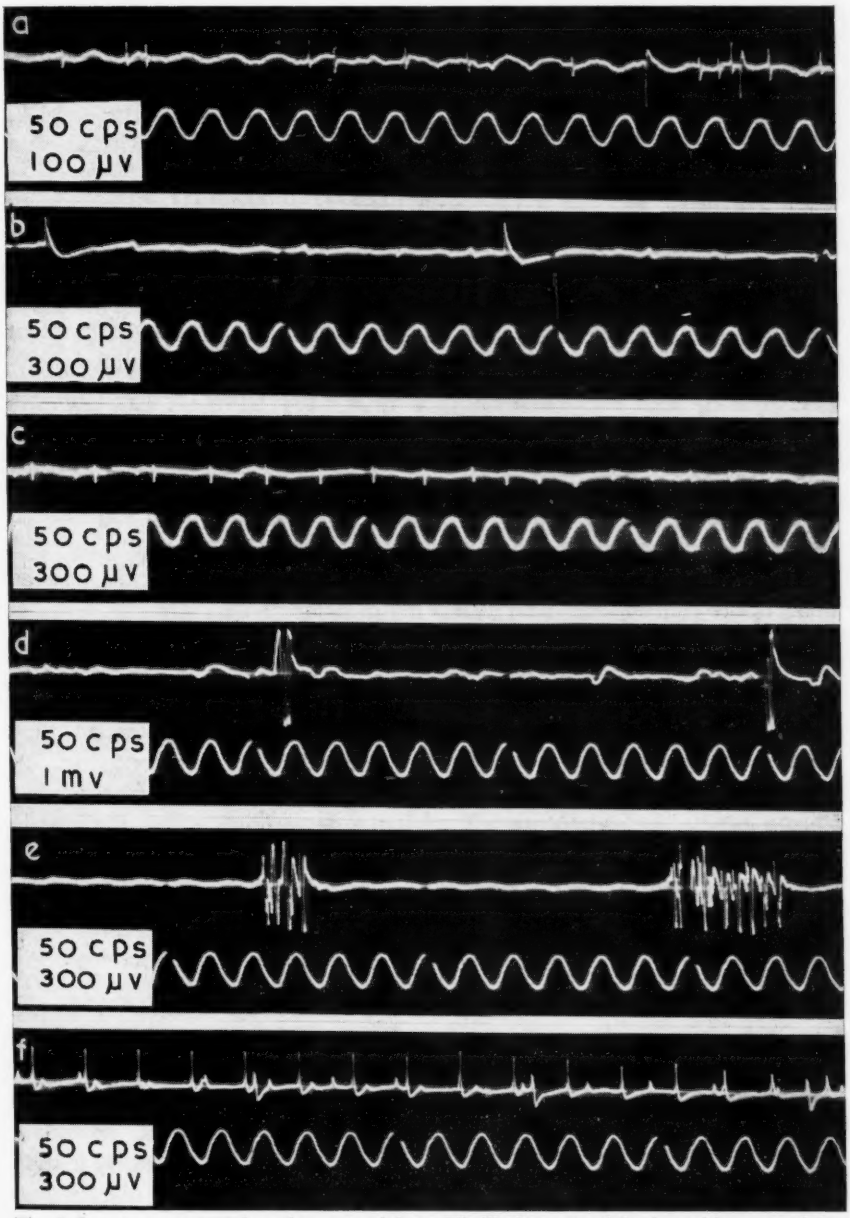

Fig. 5. Spontaneous muscle action potentials. *a*, Fibrillation potentials; *b*, positive potentials; *c*, nerve volley; *d*, fasciculation potentials; *e*, grouped (repetitive) discharges; *f*, high-frequency discharges. Calibration as indicated.

has been stimulated by interrupted galvanism. They are potentiated by neostigmine or one of its analogues and appear about 14–20 days after denervation at the time that denervated muscle becomes very sensitive to acetylcholine. They are by no means constant in denervation (Richardson, 1951b), and it is for this reason that intensity–duration curves must be used with electromyography if denervation is to be detected with accuracy.

Positive Potentials (*Sharp Waves*)

These consist of short initial positive deflection followed by a prolonged negative phase of some 30–50 msec. duration (Jasper and Ballem, 1939). Their frequency is more rapid than that of fibrillation potentials (in the range of 2–50 per second) and their sound is that of a dull thud. Their voltage shows remarkable variation. If studied, their relation to fibrillation potentials appears to be that of a decayed form of fibrillation potential.

This type of potential appears commonly during denervation and rarely in myopathies (Kugelberg and Petersen, 1949). They are not obtained from normal muscle, and therefore are a useful indication of pathological muscle. They are sometimes referred to as V waves; and, while they are readily detected by their characteristic shape, which is most constant (Fig. 5*b*), care must be taken not to confuse them with distant motor-unit activity.

Nerve Potentials

The voltage of these potentials varies from 50 to 200 μV, and their duration from 1 to 2 msec.; and, while often confused with fibrillation potentials, they are easily distinguished by their higher frequency of 50–100 per second (Fig. 5*c*). They are of no pathological significance. These potentials may be produced when a needle electrode contacts discharging nerve fibres within the muscle substance, therefore they are most commonly found in the region of the motor point (Jones, Lambert, and Sayre, 1955).

Fasciculation Potentials

In general, these potentials are of the size of large motor units and polyphasic; their irregular frequency is about 5–20 per minute (Fig. 5*d*). They are derived from the spontaneous contraction of motor units. Like fibrillation potentials, they are most easily recognized by their sound—that is, slow irregular thuds; they cannot be abolished by relaxation or contraction of an antagonist. They are sometimes referred to as slow sporadic potentials (Hirschberg and Abramson, 1950), and occur particularly in progressive lesions of the anterior horn cells (myelopathies) such as motor neurone disease, but may also occur in response to peripheral-nerve stimulation.

Their origin in motor-cell disease would appear to be in the peripheral portion of the nerve or in the myoneural junction. Thus, it has been shown that they continue after section of the motor nerve (Forster, Borkowski, and Alpers, 1946). The fact that impulses arising in these distal portions of the motor neurone can give rise to almost synchronous contraction in the muscle fibres can be explained by invoking an axon reflex. Such a phenomenon has been demonstrated by Masland and Wigton (1940). Evidence for their origin from the end-plates derives from their potentiation by neostigmine.

Grouped (Repetitive) Motor-unit Discharges

These consist of the regular activity of grouped (repetitive) discharges of motor-unit potentials. Their sound and appearance (Fig. 5*e*) are very characteristic, and they are to be found in cases of nerve irritation from pressure (e.g. prolapsed intervertebral disks, cervical rib, carpal tunnel syndrome, etc.), in facial myoclonus and such biochemical disturbances as tetany (hypocalcaemia). They are also the electromyographic pattern of benign myokymia (Denny-Brown and Foley, 1948). They are very inconstant in nerve-root pressure lesions and consequently of little value for locating such lesions; this is in contrast to the finding with fibrillation potentials, which can be useful in this way (Shea, Woods, and Werden, 1950). They are even less frequently seen in cases of progressive muscular atrophy, polyneuritis, and poliomyelitis, and in normal muscle, when their groupings are often large (1–200). These large groupings, which are similar to high-frequency discharges and occur at a frequency of about 10–15 per second, may be classified under that heading. As such they are sometimes referred to as fast rhythmic fasciculations, and their association with cramps is noteworthy.

The exact mechanism of the production of grouped motor-unit discharges is not fully understood, but they appear to be due to spontaneous nerve impulses arising in the proximal part of the longest lower motor neurones, in some cases in areas of ischaemia (Kugelberg, 1946). In this event they can be correlated with hyperexcitability (low rheobase) and accommodation breakdown (Kugelberg, 1944) and can be augmented by hyperventilation.

High-frequency Discharges

These consist of protracted trains of oscillations which occur in a variety of forms (Fig. 5*f*). Some have dimensions comparable with single-fibre potentials, others resemble motor-unit potentials, and some are bizarre in form. They are remarkable for their change in pitch, which makes them sound like diving aeroplanes. Their frequency may be 100–150 per second, dropping down to

10–20 per second (Buchthal and Clemmesen, 1941), and the dramatic quality of the sound caused by this phenomenon has led to the terms "dive-bomber" and "quasi-musical". They occur in profusion in myotonia congenita (Thomsen's disease) and dystrophia myotonica, which also exhibit a characteristic post-volitional fibrillary after-discharge.

In addition to the spontaneous activity produced by lesions of the motor unit, rhythmic patterns of motor-unit activity may be found in the involuntary movements characteristic of some diseases of the central nervous system. Although such tremors are difficult to differentiate by electromyography, the alternate bursts of interference pattern (clonic tremor) in opposing muscles in Parkinson's syndrome are characteristic and distinct from the faster tremor, generally synchronous, in opposing muscles found in anxiety states (Clare and Bishop, 1949).

Muscle twitches of apparent spontaneity may occur in cross-reinnervation —for example, following brachial plexus lesions, when motor-unit potentials with a respiratory rhythm (from the phrenic roots) may be detected in biceps and brachioradialis muscles and in the cross-reinnervation following facial palsies.

SIZE OF MOTOR-UNIT POTENTIALS ON MINIMAL VOLITION

At least four forms of motor-unit potentials may be differentiated at the stage of minimal volition; they are those of normal dimensions, highly polyphasic long-duration potentials, large-amplitude motor-unit potentials, and short-duration "broken-up" motor-unit potentials.

Normal Motor-unit Potentials

The characteristics of normal motor-unit potentials have already been discussed. Their average duration in limb muscle is generally accepted as 5 – 10 msec. (Weddell, Feinstein, and Pattle, 1944) (Fig. 6*a*). Petersen and Kugelberg (1949) gave an average duration of 7 msec. for motor-unit potentials obtained from the interossei and biceps muscles, and in facial muscles an average of 2 msec. In clinical practice amplitudes are considered only within wide limits because of their great variation produced by needle positioning, compared with the slight variations in duration. In general, the needle electrode should be positioned so as to obtain the maximal amplitude and minimal duration of the potentials.

Large Motor-unit Potentials

The presence of motor-unit potentials of large amplitude (3–5 mV) and greater duration than normal (10–15 msec.) initiating volition has been noted by

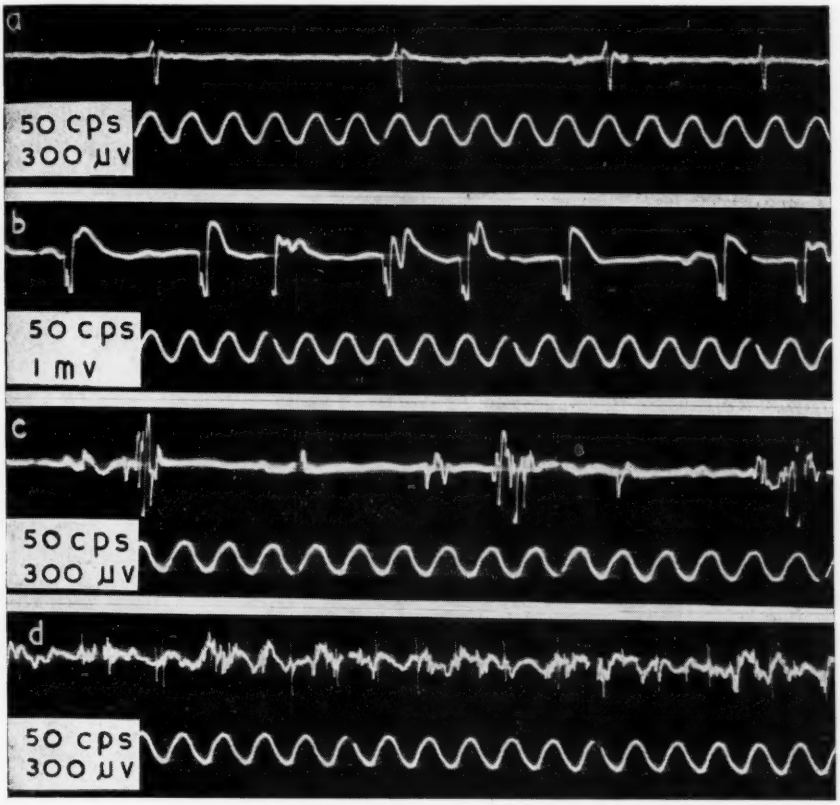

Fig. 6. Muscle potentials on minimal volition. *a*, Normal motor-unit action potentials; *b*, large motor-unit potentials; *c*, highly polyphasic long-duration potentials; *d*, short-duration "broken-up" motor-unit potentials. Calibration as indicated.

several workers in cases of neurogenic atrophy (Kugelberg, 1949; Buchthal and Pinelli, 1953a) (Fig. 6*b*). This change in both amplitude and duration and the finding of increased numbers of polyphasic motor-unit potentials are particularly marked in myelopathic lesions and may be used in their diagnosis. In contrast, peripheral neuropathies may produce an increase in the duration of motor-unit potentials (e.g. long-duration polyphasic) but not in their amplitude.

The phenomenon of synchronization of motor-unit activity in myelopathies (Buchthal and Clemmesen, 1943) is undoubtedly related to the presence of these large units. Denny-Brown (1944) compared the mechanical and the

electrical response of motor-unit activity and deduced that the phenomenon of synchronization was due to the detection by two or more needle electrodes of the same, often large, motor-unit potential. He felt that this single large motor-unit potential could be detected because of the uncovering of its activity by loss of the smaller motor-unit potentials. These normally initiate volition and summate with the large motor-unit potentials to form the normal interference pattern. Kugelberg and Taverner (1950) showed that synchronization can be produced on stimulation of the motor nerve outside the cord. Richardson (1951a), by measuring axon reflex times, produced evidence to show that two potential sources, which he demonstrated in synchronization, could be explained by the presence of branched motor units.

Highly Polyphasic Long-duration Potentials

These potentials are of low amplitude (up to 500 μV) and long duration (about 15 msec.) (Fig. 6*c*). They occur in reinnervation, the characteristics of which are the appearance of increasing numbers of motor-unit potentials becoming more and more polyphasic and the difficulty of detecting fibrillation potentials. Polyphasic potentials can also be detected during degeneration of the lower motor neurones, but at this stage seldom reach 15 msec. duration.

However, the differentiation of degeneration and regeneration can be extremely difficult at one examination. The temporal dispersion of regenerating motor-unit potentials appears to be largely due to the retarded conduction rate in the regenerating lower motor neurone.

Short-duration (Small) "Broken-up" Motor-unit Potentials

A predominance of motor-unit action potentials of short duration and increased polyphasicity (Fig. 6*d*), sometimes referred to as disintegration of motor-unit potentials, is characteristic of myopathic lesions (Kugelberg, 1949). This change occurs in the muscular dystrophies (Pinelli and Buchthal, 1953), polymyositis (Buchthal and Pinelli, 1953b), neuromyositis and dermatomyositis (Richardson, 1956a), dystrophia myotonica (but not myotonia congenita), thyrotoxic myopathy (Sanderson and Adey, 1952), and rarely in myasthenia gravis on fatigue and periodic paralysis. These potentials, which are produced by loss of muscle-fibre activity within the motor units, are found only in advanced lesions, consequently myopathic lesions are diagnosed most often by the failure to obtain signs of denervation in wasted muscles rather than by the detection of broken-up motor-unit potentials.

It is this, the second stage of electromyographic examination, that is the most difficult. Considerable skill is required to recognize over-all changes in

the type of motor-unit potentials initiating volition. Hence the use of a frequency analyser (Fig. 7). It is particularly the determination of a significant increase in the short-duration and polyphasic motor-unit potentials that characterizes the myopathies that presents difficulties. The frequency analyser reflects this change by showing a shift to the right (to the high-frequency side)—that is, an increase of the frequency components in the 1–8 kilocycle range as compared with normal muscle or neuropathies (Fig. 7). Indeed, these components often exceed those in the 0·3–1 kilocycle range. Even more difficult is the detection of a neuromyopathy in which both normal or long-duration polyphasic potentials (as in neuropathies) and short-duration potentials (as in myopathies) occur. Frequency analysis of this pattern shows widespread frequency components sometimes with double peaking, one in the centre of the low-frequency band and another in the centre of the high-frequency band.

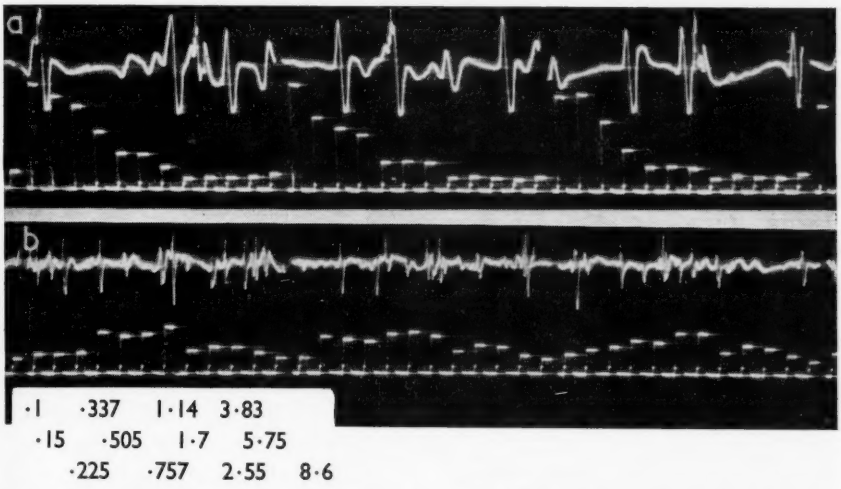

Fig. 7. Frequency analysis of motor-unit action potentials. *a*, Neuropathic lesion; *b*, myopathic lesion. Histogram frequencies indicated in kilocycles per second.

THE INTERFERENCE PATTERN PRODUCED BY MAXIMAL VOLITION

Summation of the motor-unit potentials detected by a needle electrode into the normal interference pattern on maximal volition is a criterion of normality. Lesions producing inactivity of the whole motor unit—that is, lesions situated above the point of branching of the lower motor neurone (myelopathic and neuropathic)—result in a failure to obtain this interference pattern. Lesions situated below this point (myopathic and neuromyopathic)

result in changes in the individual motor-unit potentials, but enough motor units are preserved for their potentials to summate into an interference pattern.

Findings at this stage of an electromyographic examination are conveniently classified, according to the increasing severity of the lesion, into: (1) normal interference pattern, (2) reduced interference pattern, (3) discrete or single motor-unit activity, and (4) no motor-unit activity (Fig. 8*a*, *b*, *c*).

The absence of motor-unit potentials on attempted volition is a finding of some importance in peripheral-nerve injuries. In the absence of nerve conduction and the presence of intensity-duration curves characteristic of complete denervation, it indicates complete degeneration of the nerve, although, of course, continuity of the nerve sheath may be maintained.

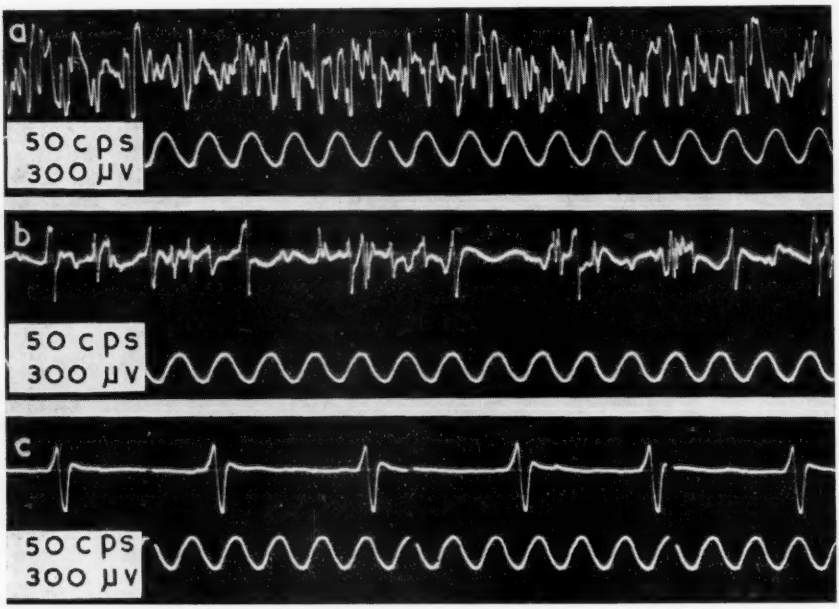

Fig. 8. Muscle-action potentials on maximum volition. *a*, Full interference pattern; *b*, reduced interference pattern; *c*, discrete motor-unit activity. Calibration as indicated.

Location of Motor-unit Lesions

All the electrodiagnostic findings described above may be used in combination to locate the part of the motor neurone which is affected by any lesion. While the various patterns are not consistent—and, indeed, in many instances

the incidence of these changes in various clinical syndromes has yet to be estimated—sufficient correlation between clinical syndromes and electrodiagnostic findings exists to permit such reports as the following:

a. IN MYELOPATHIC LESIONS

"Intensity–duration curves show sluggish responses, low (or normal to high in the presence of fibrosis) rheobases, and 100/1 msec. ratios greater than 2. Electromyograms show fibrillation and positive potentials, occasional fasciculation potentials, and on volition a reduced interference pattern of large and often polyphasic motor-unit potentials."

It is to be noted that in myelopathic lesions fibrillation potentials may be difficult to find and fasciculation potentials occasional. The most characteristic feature is the initiation of voluntary contraction by large and often polyphasic motor-unit potentials (the so-called inverted recruitment).

b. IN NEUROPATHIC LESIONS

"Intensity–duration curves show sluggish responses, low rheobases (high just before recovery and in fibrosis), and 100/1 msec. ratios greater than 2. Electromyograms show fibrillation potentials and positive potentials, and on volition a reduced interference pattern (or discrete or no motor-unit activity) of normal (or long-duration polyphasic in regeneration or degeneration) motor-unit potentials."

In earlier accounts of electrodiagnosis reference was made to distal neuropathic lesions. These are now considered to be neuromyopathic.

c. IN MYOPATHIC LESIONS

"Intensity–duration curves show brisk responses, high rheobases, and normal 100/1 msec. ratios. Electromyograms show no spontaneous activity (or high-frequency discharges in dystrophia myotonica and polymyositis), and on volition a full interference pattern of motor-unit potentials, the majority of which are of short duration and polyphasic. Frequency analysis shows a significant increase (or a shift to the right) in the high-frequency components."

d. IN NEUROMYOPATHIC LESIONS

"Intensity–duration curves show sluggish responses, high rheobases, and 100/1 msec. ratios greater than 2. Electromyograms show fibrillation and positive potentials (occasionally high-frequency discharges), and on volition a full (sometimes reduced) interference pattern of motor-unit

potentials, the majority of which are polyphasic and of both long and short duration. Frequency analysis shows a significant increase (or shift to the right or double-hump curve) in the high-frequency component."

It should be noted that partial or complete denervation cannot be judged from 100/1 msec. ratios alone, and, therefore, if electromyography is not used full curves should be recorded.

Electrodiagnosis of Fasciculation

In the absence of coexistent signs and symptoms, persistent muscle fasciculation presents a difficult diagnostic problem. It is the muscle twitches detected electromyographically as fasciculation potentials and grouped (repetitive) motor-unit potentials that are seen clinically as fasciculation (coarse fibrillation). If they occur in calf or eyelid muscles they are generally regarded as benign, but their occurrence in other muscles suggests progressive anterior-horn-cell disease and, therefore, a poor prognosis. Such a prognosis can often be reversed by careful electrodiagnostic investigation.

Fasciculation in Motor-Cell Disease

In motor-cell disease the potentials accompanying fasciculation are irregular not only in amplitude and grouping but often in their polyphasicity on repetition. The most important electrodiagnostic sign in the identification of this form of fasciculation is the coexistence of signs of denervation and of a myelopathic lesion. In general, signs of denervation appear within three months of the onset of fasciculation in motor-cell disease (Schwab, Stafford-Clark, and Prichard, 1951). The use of "tensilon" given intravenously in 10-mg. doses to initiate fasciculation is occasionally useful in diagnosis, but it will be found that many normal people fasciculate with such doses.

Cramp and Compression Fasciculation

Fasciculations occurring during cramps have been investigated by Denny-Brown and Foley (1948). They occur chiefly in the calf muscles and are more transient than other forms. They follow the general characteristic of fasciculation potentials and, like those of motor-cell disease, are irregular in rhythm and often of varying polyphasicity. Their characteristic form and their cancellation by volitional activity appear to indicate that they arise in the terminal branches of the lower motor neurone and have a changing focus of origin. Because they are not influenced by tensilon the site is unlikely to be at the myoneural junction. They are readily distinguished as a form of benign muscle fasciculation because they are not accompanied by signs of denervation.

Fasciculation following compression of nerve roots or peripheral nerves also has the general characteristic of fasciculation potentials in being spontaneous motor-unit discharges. However, these are usually of a simple diphasic form (Brazier, Watkins, and Michelson, 1950), which tends to remain constant on repetition. Their coexistence with repetitive motor-unit discharges is common. By the use of two cuffs Kugelberg and Cobb (1951) demonstrated that the point of origin of these impulses was in the proximal part of the lower motor neurone. Indeed, this experimental evidence is supported by the occurrence of their constant wave form on repetition. In these lesions accompanying signs of denervation are usually localized to muscles supplied by a single nerve root or trunk. A notable exception to this is the widespread fasciculation which may occur in cervical spondylosis but which may well be due to a true myelopathic lesion through involvement of the spinal arteries.

Benign myokymia, first described by Schultz in 1895, usually occurs in isolated muscles such as that of the calf and the small muscles of the hand, but may be widespread. The majority of cases are of unknown origin, but hereditary forms have been described, and the condition has been observed in association with thyrotoxicosis (Harman and Richardson, 1954). The twitches are seen electromyographically as grouped or repetitive motor-unit potentials in the absence of denervation.

Electrodiagnosis of Facial Palsies

The most important test in the diagnosis of facial palsies is the testing of nerve conduction. By applying a 0·3 msec. pulse to the region of the stylomastoid foramen and comparing the threshold intensities on the normal and abnormal side the presence of a degenerative lesion can be inferred. The maintenance of normal nerve conduction beyond the third day carries a good prognosis; beyond a week an excellent one. If nerve conduction disappears, the lesion must be a degenerative one and full recovery must await regeneration—that is, three to four months. The prognosis of a degenerative lesion is far worse if it is a complete lesion. Hence the retention of some motor-unit activity in the absence of nerve conduction improves the prognosis.

Electrodiagnosis of the Childhood Hypotonias

In children the usual electrodiagnostic procedure is conveniently reversed, electromyography being carried out first. In the group of childhood hypotonias (Werdnig–Hoffmann's disease, the infantile myopathies, and the amyotonia congenita syndrome) electromyography reveals either a normal motor-unit pattern, a myopathic pattern, or a neuropathic pattern characterized by reduced motor-unit activity. In the latter instance the search for fibrillation

potentials is important, and, failing this, it is necessary to carry out intensity–duration curves under an anaesthetic. Reduced motor-unit activity with fibrillation (or denervated intensity–duration curves) is characteristic of Werdnig–Hoffmann's disease, but not all cases appear to be progressive. Myopathic patterns are found excessively rarely under the age of one year, but it must be emphasized that a normal pattern may coexist with an early myopathy.

Electrodiagnosis of Myasthenia Gravis

Neither fatigue tests nor response to neostigmine or one of its analogues give constant results to allow the diagnosis of myasthenia gravis with any certainty. The most constant finding is that based on the production of a competitive inhibition block in myasthenia gravis by decamethonium iodide ("C 10"), in contrast to the production in normal patients of a depolarizing block (Churchill-Davidson and Richardson, 1953). Evidence for this derives from the fact that 2·5 mg. of decamethonium given intravenously to normal subjects produces a drop in the action potentials of the hypothenar eminence (produced in response to supramaximal stimulation of the ulnar nerve at 10 per second), and this change in action potentials is worsened with a subsequent dose of 10 mg. of tensilon. In contrast, in myasthenia gravis 2·5 mg. of decamethonium does not produce a drop in the action potential, although in the clinically weak muscles paralysis occurs on attempted volitional effort. A subsequent dose of tensilon, while not affecting the action potential of the hypothenar muscles, reverses any volitional paralysis. (It must be emphasized that the use of relaxant drugs in myasthenia gravis requires facilities for artificial respiration.)

Apart from the above standard techniques, various additional manœuvres may be used as an aid to the diagnosis of neuromuscular diseases. While details of these are beyond the scope of this article, reference must be made to two methods which are likely to find a place in routine clinical electrodiagnosis. These are the technique of recording nerve-action potentials through the skin (Dawson and Scott, 1949) and that of Magladery, Porter, Park, and Teasdall (1951), who recorded action potentials from the spinal cord and nerve roots by means of needle electrodes inserted through the vertebral interspaces and studied various two-neurone (monosynaptic) reflexes. Motor-unit activity may also be used as an index of long tract lesions, and occasionally characteristic patterns have been obtained—for example, that obtained by Richardson (1956b) in benign myalgic encephalomyelitis.

Finally, it is the wise electrodiagnostician who realizes that the most important instruments in the electrodiagnostic clinic are, in order: a pin, a patella hammer, a square-pulse stimulator, and lastly an electromyograph.

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ORIGINAL PAPER

A COMPARISON OF THE EFFECTS OF CORTISONE AND PREDNISONE IN RHEUMATOID ARTHRITIS

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THE recognition that apparently minor changes in the steroid molecule may profoundly modify both the clinical and biochemical effects of these substances has led to the trial of many variants of cortisone. Δ^1-Dehydrocortisone (prednisone; "meticorten"; "metacortandracin") has recently been reported as having an anti-inflammatory effect four or five times that of cortisone (Bunim *et al.*, 1955; Dordick and Gluck, 1955; Gray and Merrick, 1955; Nabarro *et al.*, 1955; Hart *et al.*, 1955; Villa *et al.*, 1955). Most of these reports have been confined to precise metabolic but rather indefinite clinical assessments—the latter being largely based on general impressions as to relative potency and the incidence of side-effects. Considerable experience has now been gained, however, in the techniques of measurement of clinical improvement in rheumatoid arthritis, so that, given adequate experimental methods and careful techniques of assessment, it is possible to compare the effects of such substances with only few patients and a short trial (Quin *et al.*, 1950; Mason, 1953). The information gained is, of course, limited, since it provides no data as to the effect of long-term administration or the incidence of later complications.

The present trial was designed simply to test the claim that Δ^1-dehydrocortisone is approximately four times as potent, dose for dose, as cortisone in its effects on rheumatoid arthritis.

METHOD

Eight patients with active rheumatoid arthritis who were thought to have sufficient reversible features to enable therapeutic effects to be measured were admitted to hospital. A period of bed rest was first given, during which the condition of the patient was assessed at frequent intervals. When two or three assessments became almost identical the trial was begun. Details of the cases, with erythrocyte sedimentation rates (E.S.R.) on admission, are given in Table I.

Both drugs were administered orally in divided doses. Cortisone was given in a dose of 200 mg. the first day and 100 mg. daily thereafter for nine days;

the drug was then gradually withdrawn over the next four days. Prednisone was given in a similar manner but in one-quarter the dose—i.e. 50 mg. on the first day followed by 25 mg. daily for nine days and then gradually reduced. The order of trial is shown in Table II.

The following features were measured: E.S.R. (Westergren); joint swelling by means of a ring test (Hart and Clark, 1953); spontaneous pain, stiffness, and subjective improvement, by an arbitrary method of assessment; joint tenderness; joint range; strength of grip; together with a series of function tests—not less than three in each case—and estimation of urinary excretion of 17-ketosteroids and 17-ketogenic steroids. The tests for a given patient were identical in Parts I and II of the trial.

TABLE I

DETAILS OF CASES

Case No.	Sex	Age (years)	Duration of Disease (years)	E.S.R. on Admission (mm. per hour; Westergren)
1	F	43	10	43
2	F	35	5	83
3	F	41	16	17
4	M	60	4	15
5	F	54	9	62
6	F	41	3	10
7	F	59	3	25
8	F	28	15	93

Each patient was subjected to assessment on admission and then every few days until two consecutive assessments gave similar results. Part I of the trial was then started and a reassessment made after ten days' therapy. At least a fortnight was allowed to elapse until the results of two assessments became similar in the "relapse" interval. Part II of the trial was then begun, and a final assessment was made at the end of the second ten-day period of therapy. Each assessment was carried out at the same time during the day—namely, 9.30 a.m. —to avoid fluctuation due to morning stiffness, etc.

RESULTS

The clinical response is shown in Table II. The results of the tests are given in the following pages.

TABLE II

ORDER OF TRIAL AND CLINICAL RESPONSE

Case No.	Order of Trial	Clinical Response and Remarks
1	Prednisone/cortisone	Considerably improved while on prednisone; response to cortisone less dramatic. Preferred former
2	Cortisone/prednisone	Cortisone disappointing; much better on prednisone
3	Prednisone	Developed gastric pain on eighth day; drug stopped. Despite negative barium meal examination, further hormone therapy postponed. Arthritis responded poorly
4	Prednisone/cortisone	Intelligent patient, easy to assess objectively and subjectively. Responded to both drugs, but preferred cortisone
5	Cortisone/prednisone	Difficult to assess subjectively. Suggestion of fluid retention, confirmed by rise in weight while on cortisone
6	Cortisone/prednisone	Moderately good response to both
7	Cortisone/prednisone	Difficult to assess subjectively; objectively about equal improvement each day. Functionally a little better on prednisone. Severe relapse between Parts I and II of trial, possibly due to receipt of news of serious road accident to husband at end of Part I
8	Prednisone/cortisone	Co-operative and intelligent. Slightly better on prednisone

ERYTHROCYTE SEDIMENTATION RATE (Table III)

The mean E.S.R. before prednisone treatment was started was 37 mm. per hour (Westergren), and before cortisone treatment 40 mm. per hour. It fell to a mean of 22 mm. per hour during prednisone administration, and to a mean

of 24 mm. while cortisone was being given. Both these falls are statistically significant, but there is no statistical evidence that the fall produced by cortisone is greater than that due to prednisone.

TABLE III

EFFECT OF TREATMENT ON ERYTHROCYTE SEDIMENTATION RATE
(mm. per hour)

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	43	15	−28	15	12	− 3
2	55	50	− 5	100	55	−45
3	17	6	−11	—	—	—
4	30	15	−15	42	15	−27
5	34	2	−32	40	6	−34
6	12	2	−10	10	4	− 6
7	15	5	−10	18	6	−12
8	92	24	−68	57	16	−41
Total	298	119	−179	282	114	−168
Mean	37·3	14·9	−22·4	40·3	16·3	−24·0

Mean E.S.R. before Part I, 44·8 mm. per hour; before Part II, 32·9 mm. per hour. Difference, 11·9 mm. per hour.

For prednisone $t=3{\cdot}190$ $P<0{\cdot}05$; for cortisone $t=3{\cdot}718$ $P<0{\cdot}05$. Difference prednisone/cortisone $t=0{\cdot}16$ $P>0{\cdot}05$.

RING SIZE

Results of this test are given in Table IV. The most active proximal interphalangeal joint was chosen in each case and the swelling measured by sizing with jewellers' rings. There was a statistically significant improvement in the swelling with each drug, but no significant difference in effect between the two.

SPONTANEOUS PAIN, STIFFNESS, AND SUBJECTIVE IMPROVEMENT (Table V)

Assessment of these factors was intended to provide an approximate measure of "how the patient felt". The patient was asked each day, both before and during treatment, whether he had any stiffness or pain in two or three selected active joints; if so, the degree of this was assessed from the duration

TABLE IV

CHANGE IN RING SIZE DURING TREATMENT

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	61	58	− 3	62	60	− 2
2	64	62	− 2	63	63	0
3	60	56	− 4	—	—	—
4	64	60	− 4	62	60	− 2
5	67	61	− 6	64	61	− 3
6	55	54	− 1	57	54	− 3
7	61	61	0	63	61	− 2
8	58	53	− 5	56	54	− 2
Total	490	463	−25	427	413	−14
Mean	61·3	58·3	−3·2	61·0	59·0	−2·0

Mean ring size before Part I, 61·3; before Part II, 61·0. Difference, 0·3.

For prednisone $t=4{\cdot}417$ $P<0{\cdot}05$; for cortisone $t=5{\cdot}054$ $P<0{\cdot}05$. Difference prednisone/cortisone $t=1{\cdot}345$ $P>0{\cdot}05$.

TABLE V

SUBJECTIVE IMPROVEMENT

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	7	1	− 6	4	1	− 3
2	8	4	− 4	9	4	− 5
3	10	4	− 6	—	—	—
4	4	2	− 2	5	2	− 3
5	11	1	−10	8	1	− 7
6	10	1	− 9	10	3	− 7
7	13	1	−12	14	7	− 7
8	13	2	−11	8	3	− 5
Total	76	16	−60	58	21	−37
Mean	9·5	2·0	−7·5	8·3	3·0	−5·3

Mean subjective state before Part I, 9·3; before Part II, 8·4. Difference, 0·9.

For prednisone $t=5{\cdot}976$ $P<0{\cdot}05$; for cortisone, $t=7{\cdot}714$ $P<0{\cdot}05$. Difference prednisone/cortisone, $t=1{\cdot}479$ $P>0{\cdot}05$.

of each episode as follows: no pain—no points; slight pain in early morning—1 point; more severe pain lasting several hours—2 points; pain lasting all day—3 points; pain lasting day and night—4 points.

It will be seen that there is a significant statistical improvement with both drugs, but no significant difference between each.

Joint Tenderness (Table VI)

In every case six joints were examined at each assessment for tenderness, and this was graded from 1 (the mildest) to 4 (the most severe).

Statistical calculation reveals that there is a significant improvement with prednisone but not with cortisone, although if the series was bigger improvement with cortisone would probably be apparent. There is, however, no significant difference in the results obtained with prednisone and cortisone in this test.

TABLE VI

Comparison of Joint Tenderness

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	15	5	−10	12	12	0
2	13	5	− 8	12	10	− 2
3	15	16	+ 1	—	—	—
4	12	6	− 6	12	5	− 7
5	13	7	− 6	12	10	− 2
6	16	3	−13	15	6	− 9
7	18	12	− 6	16	14	− 2
8	16	5	−11	15	6	− 9
Total	118	59	−59	94	63	−31
Mean	14·8	7·4	−7·4	13·4	9·0	−4·4

Mean joint tenderness before Part I, 14·2; before Part II, 14·1. Difference, 0·1.

For prednisone, $t=5{\cdot}332$ $P<0{\cdot}05$; for cortisone, $t=2{\cdot}028$ $P<0{\cdot}05$. Difference prednisone/cortisone, $t=1{\cdot}479$ $P>0{\cdot}05$.

Joint Range (Table VII)

In each case four joints were selected which appeared active and had a limited range of movement. Joint range is difficult to assess, and therefore it

was decided to divide the possible range into four grades: Grade 4, completely stiff to 25% range—4 points; Grade 3, 25%–50% range—3 points; Grade 2, 50%–75%—2 points; Grade 1, 75%–100%—1 point. In each part of the trial this test showed significant improvement, but there was no significant difference between Parts I and II.

TABLE VII

COMPARISON OF CHANGES IN JOINT RANGE

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	10	4	− 6	7	3	− 4
2	10	7	− 3	11	8	− 3
3	6	3	− 3	—	—	—
4	7	3	− 4	5	3	− 2
5	7	5	− 2	9	5	− 4
6	6	1	− 5	7	3	− 4
7	9	7	− 2	10	7	− 3
8	10	6	− 4	9	6	− 3
Total	65	36	−29	58	35	−23
Mean	8·2	4·5	−3·6	8·3	5·0	−3·3

Mean joint range before Part I, 8·8; before Part II, 7·6. Difference, 1·2.

For prednisone, $t=7{\cdot}235$ $P<0{\cdot}05$; for cortisone, $t=36{\cdot}58$ $P<0{\cdot}05$. Difference prednisone/cortisone, $t=0{\cdot}468$ $P>0{\cdot}05$.

GRIP TEST (Table VIII)

In each case the grip of both hands was measured by the patient gripping a sphygmomanometer cuff, inflated to 30 mm. mercury, which had been permanently folded and pinned. Three readings for each hand were taken on each occasion and the mean calculated.

It was found difficult to achieve a full relapse between Parts I and II of the trial. There was significant improvement statistically only with prednisone, but clinically both drugs appeared to produce improvement.

FUNCTION

An identical series of tests of function were carried out with both prednisone- and cortisone-treated patients, points being awarded for the time taken for each test.

TABLE VIII

COMPARISON OF VARIATION OF GRIP DURING TREATMENT

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	34	65	+31	44	44	0
2	40	45	+ 5	15	36	+21
3	42	60	+18	—	—	—
4	120	240	+120	200	285	+85
5	160	230	+70	50	245	+195
6	50	200	+150	55	175	+120
7	30	30	0	24	43	+19
8	45	65	+25	50	70	+20
Total	521	935	+419	438	898	+460
Mean	65·1	116·9	+52·4	62·6	128·3	+65·6

Mean grip before Part I, 48·2; before Part II, 71·8. Difference, −23·6.

For prednisone, $t=2{\cdot}729$ $P<0{\cdot}05$; for cortisone, $t=2{\cdot}430$ $P>0{\cdot}05$. Difference prednisone/cortisone, $t=0{\cdot}406$ $P>0{\cdot}05$.

TABLE IX

EFFECT OF CORTISONE AND PREDNISONE THERAPY ON FUNCTION

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	8	6	+ 2	6	3	+ 3
2	8	7	+ 1	10	6	+ 4
3	7	8	− 1	—	—	—
4	7	4	+ 3	6	4	+ 2
5	7	5	+ 2	8	5	+ 3
6	8	3	+ 5	9	5	+ 4
7	11	7	+ 4	12	11	+ 1
8	10	8	+ 2	10	6	+ 4
Total	66	48	+18	61	40	+21
Mean	8·3	6·0	+2·3	8·6	5·7	+3·0

Mean function before Part I, 8·8; before Part II, 8·0. Difference, 0·8.

For prednisone, $t=4{\cdot}859$ $P<0{\cdot}05$; for cortisone, $t=6{\cdot}882$ $P<0{\cdot}05$. Difference cortisone/prednisone, $t=0{\cdot}906$ $P>0{\cdot}05$.

Both drugs produced significant improvement, but one was not significantly better than the other. The mean difference between Parts I and II of the trial was very small (0·8, or less than 10%), indicating the accuracy of the method. Table IX shows the full results.

Urinary 17-Ketosteroids and 17-Ketogenic Steroids

These estimations showed variable results. The excretion of 17-ketosteroids was increased by both prednisone and cortisone, although with the latter the amount was not statistically significant. The estimations of 17-ketogenic steroids had an even wider variation and none of the statistical calculations showed any significant variable. The full results are given in Tables X and XI.

TABLE X

Variations in Urinary 17-Ketosteroid Excretion
(mg. in 24 hours)

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	8·5	4·6	−3·9	9·9	9·1	−0·8
2	10·0	6·5	−3·5	7·0	9·7	+2·7
3	9·6	7·9	−1·7	—	—	—
4	13·0	12·0	−1·0	12·0	23·0	+11·0
5	15·0	9·3	−5·7	14·0	21·0	+7·0
6	14·0	12·0	−2·0	13·0	8·0	−5·0
7	5·2	3·9	−1·3	12·5	5·0	−7·5
8	5·9	5·1	−0·8	23·0	6·6	−16·4
Total	81·2	61·2	−19·9	91·4	82·4	−9·7
Mean	10·2	7·7	−2·5	13·1	11·8	−1·4

Mean before Part I, 10·4 mg. in 24 hours; before Part II, 13·0 mg. in 24 hours. Difference, 2·6 mg. in 24 hours.

For prednisone, $t=2{\cdot}365$ $P<0{\cdot}05$; for cortisone, $t=0{\cdot}475$ $P>0{\cdot}05$. Difference cortisone/prednisone, $t=0{\cdot}332$ $P>0{\cdot}05$.

DISCUSSION

Judged by the uniformity of the results obtained, all the tests used in the assessment can be considered to be moderately accurate. Apart from estimation of urinary ketosteroid excretion during cortisone therapy, all tests except that

TABLE XI

VARIATION IN URINARY KETOGENIC STEROID EXCRETION
(mg. in 24 hours)

Case No.	Prednisone Treatment			Cortisone Treatment		
	Before	During	Change	Before	During	Change
1	11·5	7·3	−4·2	28·0	24·0	−4·0
2	10·0	11·0	−11·0	5·0	19·0	+14·0
3	7·4	10·0	+2·6	—	—	—
4	22·0	17·0	−5·0	17·0	34·0	+17·0
5	27·0	12·5	−14·5	9·0	40·0	+31·0
6	19·5	18·0	−1·5	19·5	27·0	+7·5
7	16·5	14·0	−2·5	19·5	14·5	−5·0
8	—	—	—	23·0	19·0	−4·0
Total	113·9	89·8	−24·3	121·0	177·5	+56·5
Mean	16·3	12·8	−3·5	17·3	25·3	+8·1

Mean before Part I, 10·6 mg. in 24 hours; before Part II, 20·1 mg. in 24 hours. Difference, 9·5 mg. in 24 hours.

For cortisone, $t=2·010$ $P>0·05$; for prednisone, $t=1·946$ $P>0·05$. Difference cortisone/prednisone, $t=2·046$ $P>0·05$.

of joint tenderness and the grip test showed a statistically significant improvement. However, throughout the trial there was never any evidence to suggest that prednisone in the dosage employed (i.e. one-quarter that of cortisone) was more or was less effective than cortisone. The relapse was good between Parts I and II of the trial, as shown by the difference of the means in each test.

It is concluded that there is no determinable difference, clinically or as the result of laboratory investigation, showing that prednisone is superior or inferior to four times the amount of cortisone as used in this trial. This is regarded as being in conformity with the claim that prednisone is four times as effective as cortisone in an equal dosage.

Side-effects were few with both drugs in this small trial.

SUMMARY

To test the claim that prednisone is a more effective anti-inflammatory agent than cortisone, both drugs were given in separate trials to eight patients with rheumatoid arthritis and the results compared. The response was assessed

clinically and by a number of tests, and the results were analysed statistically. In general they appear to confirm that prednisone is four times as potent as cortisone in an equal dosage.

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ORIGINAL PAPER

TENNIS ELBOW: A NEW CONCEPTION

BY L. C. L. GONET

From the Orthopaedic Department, West Middlesex Hospital, Isleworth

TENNIS elbow was first described in 1896 by Bernhardt, who called it epicondylitis humeri. The aetiology of the condition is still far from clear, however. Cyriax in 1936, reviewing ninety articles on the subject, found fourteen or more different causes. In 1937 Mills described his method of manipulation based on the presence of nipped synovial fringes, a view put forward earlier by Trethowan. Osgood regarded the condition as a bursitis; but in a very full account Lahz, in 1947, denied the presence of bursae and suggested a synovitis in the radiohumeral joint proceeding to fibrosis as the possible cause. Mercer (1950), Watson-Jones (1943), and Furlong (1954), among many others, favour trauma as the exciting factor. Furlong states that there is always to be found a degeneration of the musculo-tendinous origin of the extensors of the forearm. Recently strain of the orbicular ligament has been added to the long list by Bosworth (1955) of New York. The Germans consider malnutrition and hypovitaminosis, which were rife in their country in the immediate post-war years, together with unaccustomed manual labour, important factors in the aetiology of the condition, thus emphasizing its more general aspects.

In the main, however, there are two schools of thought: those who favour an inflammatory origin, as the name external epicondylitis would suggest, and those who favour a traumatic cause. Both schools treat the condition in the same way and both acknowledge its intractability.

The purpose of this paper is to show the relationship between a cervical vertebral disk type of lesion and tennis elbow, and to encourage clinicians to take a broader view of what has hitherto been regarded as a local lesion. The evidence is purely clinical and radiological, based on observation over the past two years and a review of all cases in the physiotherapy and out-patient departments of Heatherwood Orthopaedic Hospital, the Windsor Group of Hospitals, and West Middlesex Hospital, Isleworth. A follow-up study of 105 such cases has provided enough data to suggest a different conception of the condition. Thus 87% had radiologically demonstrable cervical vertebral pathology; 60·5% had definite hypoaesthesia in the area of the dermatome affected; 71% of the patients were women between the ages of 29 and 55, and except for two cases the men were in the same age group.

BASIS OF THE THEORY

Furlong states that any case in which there is pain in the shoulder or in the wrist as well as in the elbow is not one of tennis elbow, and that was the criterion used in the present series.

The sixth cervical dermatome is principally affected; it may be overlapped by the fifth cervical dermatome. The motor supply to the muscle group which includes the brachio-radialis comes from the anterior of root C.6, and the same root is involved, but to a lesser extent, in the motor nerve supply of the extensor carpi radialis longus and brevis. Therefore a lesion in the region of the 6th cervical vertebra or of the overlying soft tissue should produce clinical features related to the area of the extensor origin and, being a segmental lesion, should affect the reflexes, muscle power, and sensitivity.

Reflexes

There was no conclusive evidence that the reflexes were sufficiently abnormal to be of any significance, but many of the cases, though at first clinical examples of true tennis elbow, when seen a year or more later had developed unmistaken features of cervical root irritation, and then the picture had changed. This is an important point in the theory.

Muscle Power

There was no muscle weakness in the acute stage of the condition. Patients dropped relatively light objects because of the excruciatingly sharp pain at the elbow as soon as they tried to grip. Later on there was not so much pain as a feeling of weakness. Clinically, it is not easy to demonstrate this weakness, but a muscle test similar to the Ritchie–Sneath test would be more accurate, while it is possible almost to pinpoint the central lesion by electromyography. There is often a "prominence" in the region of the external epicondyle, which is remarked on by the patient. This is due to relative wasting of the extensor origin as compared with the normal arm, bringing into sharper relief the epicondyle, much as a knee looks bigger when there is quadriceps wasting.

Sensory Effects

There is local pain, both subjective and on deep palpation, with alteration in the sensitivity of the skin to pin-prick. Hypoaesthesia along the outer side of the forearm was present to a greater or less extent in 60·5% of the patients. The dermatome corresponds to that supplied by C.6, but in true tennis elbow the hypoaesthesia never extended below the level of the radial styloid. It was most marked over the area between the brachio-radialis and extensor digitorum

communis, sometimes directly over the external epicondyle or even more proximally in the lower reaches of the 5th cervical dermatome. It is significant that sometimes a local injection of procaine into the extensor origin produces pain almost exactly in the area outlined by the 6th cervical dermatome. Sensitivity was judged by pin-prick, but precautions were taken to avoid fallacious results. Bearing in mind that the skin on the back of the forearm is normally slightly less sensitive, the hypoaesthesia was assessed in comparison with the unaffected arm. Account was also taken of the fact that some individuals have a higher threshold of sensitivity to pin-prick.

The deep pain accurately localized to the area concerned is a feature of the condition. The external epicondyle is tender on sustained deep pressure in normal subjects, but (reversing the clinical approach to the problem) is much more so in the typical cervical root syndrome. It is possible for pain fibres in muscle to become more sensitive from a central lesion, but further clinical and physiological data are required before the mechanism of muscle pain can be properly elucidated. Compression of these sensitized nerve-endings in the region of the extensor origin during contraction will cause the local pain which is such a typical feature of the condition, and this would occur during the resisted pronation and supination movement of wringing out clothes, filling a hot-water bottle, or making the back and forehand strokes of tennis.

The local tenderness of tennis elbow can be explained by analogy with the painful areas that are found in lumbar disk prolapse. In motor nerves 40% of the fibres are afferent, and some subserve conscious sensation of deep pressure, pressure pain, etc., and if sensitized would account for the tenderness on palpation. The autonomic system also plays a part in the mechanism of the local pain. The elbow region has a rich sympathetic nerve supply; the afferent fibres of these nerves must eventually pass into the posterior nerve roots, which have a large proportion of non-medullated fibres in their structure and are readily stimulated. The persistence of the pain and the periostitis seen radiologically may be due to the "prominence" of the epicondyle as explained above, making it more liable to repeated knocks when otherwise it would be cushioned.

Radiological Appearances

The X-ray findings have been startling. Loss of disk space, osteoarthritis with anterior and posterior lipping, spondylolisthesis, and narrowing of the intervertebral foramina have all been observed in the C.5–C.6 area in 87% of cases. One case had bilateral cervical ribs, and one had large C.7 transverse processes. It may be that a similar series of cases, in the same age group, without tennis elbow would show as high a percentage of cervical vertebral

pathology, but the clinical coincidence, especially since many of the patients followed up have developed a true cervical root syndrome, is difficult to ignore.

TREATMENT

The average duration of treatment in the physiotherapy department was 11 weeks. No case was wholly symptom-free even a year after discharge, and, as mentioned above, many patients were under treatment later for a cervical disk lesion.

It is probable that treatment directed to the central lesion from the start may reduce the time needed and have more satisfactory results. Leriche in 1936 reported the astonishing results of local procaine infiltration in tennis players. Tavernier (1946) described sensory denervation of this area. Local injection of hydrocortisone and hyaluronidase is the most favoured form of therapy at the moment.

As this paper is restricted to a clinical review and is not directly concerned with treatment, I will merely mention that treatment directed to the neck and shoulder girdle alone *ab initio* has been successful. In intractable cases perhaps a sympathetic block may be the answer.

So far as treatment is concerned, however, this report is to be regarded only as a preliminary communication. It is to be hoped that it goes some way towards correcting the statement of Kohlmayer (1952) that no author has properly explained "the classification of the pathogenesis, aetiology, and therapy of this annoying ailment".

SUMMARY

Attention is drawn to the frequent clinical association between tennis elbow and a cervical root syndrome. A follow-up of 105 cases of tennis elbow has shown X-ray evidence of cervical vertebral pathology in 87% of cases.

It is suggested that the symptom-complex called tennis elbow has its origin in a central root lesion and is not due to a local lesion at the elbow as previously described.

ACKNOWLEDGMENTS

I am indebted to the Consultant Orthopaedic Surgeons, Mr. G. P. Arden, Mr. W. Herschell, Mr. E. Devenish, and the late Mr. L. A. Key, for permission to use their cases; to Professor Last for his help with the anatomical problems; and to the Departments of Physical Medicine and Radiography of the Windsor Group of Hospitals, and West Middlesex Hospital, Isleworth, for their co-operation.

The list of references appears on page 69

A CHAIR FOR THE STIFF-LEGGED PATIENT

THE chair illustrated opposite enables a patient with stiff legs to attain the erect position with the minimum of effort by using a small hydraulic jack. As can be seen in the photographs, the operating handle is within easy reach and is simple to operate. The seat rises and tilts forwards, pushing the patient on to his feet. Two handles are provided for steadying while adjusting sticks or crutches. The height can be adjusted by distance pieces slipped into each leg of the chair. The chair is made of tubular steel and is inexpensive. It originated in the Physical Medicine Department at Black Notley Hospital, Braintree, Essex, the prototype being made in the hospital works department. It is now being manufactured by Stanley Cox Ltd.

ACKNOWLEDGMENT

My thanks are due to Mr. L. A. Haug, M.C.S.P., for inventing the chair, and the Colchester Hospital Management Committee for providing the prototype.

K. W. NICHOLLS PALMER

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PLATE I

K.W.N.P. facing page 74]

REVIEWS OF BOOKS

PHYSICAL MEASURES IN THE TREATMENT OF POLIOMYELITIS. By R. J. S. Reynolds. *Pp. 140. 12s. 6d. London: Faber and Faber. 1956.*

This book is written by a physiotherapist who during the past nine years has superintended the treatment of a large number of patients suffering from poliomyelitis. He has thus, he says, been able to observe and assess the value of various methods used throughout the country, and this has convinced him of the superior value of the particular routine of treatment used at Queen Mary's Hospital for Children at Carshalton. The immediate aim is, of course, to save life in the acute and post-acute phases of the illness; after this the real work of promoting recovery of function by exercises is the obvious aim.

The principle of the physical methods described in this book is based on the concept of Miss Elizabeth Kenny, which, to say the least, is controversial and has no proven physiological basis. It is difficult to see quite how the author has reached the conclusion that the advocated routine of treatment is the one of choice, although he makes the statement that he has "been able to compare the results of various methods of treatment through observation of our own cases, and those admitted after initial treatment elsewhere". Apparently the comparison is based on 600 patients treated, of whom only a few have been admitted in the late convalescent stage from other hospitals for further rehabilitation. Exact figures are not given, nor are the methods of assessing improvement described.

During the last ten years considerable interest has been taken in the investigation and treatment of poliomyelitis, and there is little doubt that all methods in use to-day have been carefully considered and investigated. One is forced, therefore, to conclude that the methods advocated in this book do not provide any greater therapeutic efficiency than up-to-date orthodox methods. At least one disadvantage of the methods described is the need for expensive and cumbersome apparatus.

W. RUSSELL GRANT

POSTURAL DRAINAGE. By E. Winifred Thacker, M.C.S.P. *Pp. 56. 8s. 6d. London: Lloyd-Luke. 1956.*

The physiotherapist is now acknowledged to be a vital member of the modern chest unit, and in this small booklet Miss Winifred Thacker shows why this is so. The correct performance of the procedures described here may well determine whether or not a successful outcome is achieved—a view endorsed by those best qualified to judge, including no less an authority than Mr. Holmes Sellors, who contributes the foreword. It is often difficult to win the very necessary confidence of these patients, and Miss Thacker does well continually to stress the importance of a firm yet sympathetic approach.

An excellent feature of this book is the presentation of photographs of postural drainage technique cheek by jowl with a diagram of the relevant part of the bronchial tree, thus clearly emphasizing the importance of careful positioning. The diagrams and photographic illustrations are simple and of a high standard of reproduction, though it is a pity that there is not more cross-reference to the illustrations from the text. In a work dealing essentially with techniques a great deal of irritation may be

caused by setting illustrations several pages away from the main text relating to them, and the captions are, on the whole, not detailed enough to bridge the gap.

Somewhat curious concepts are hinted at in alluding to the relationship between anxiety and muscular relaxation, and to the physiological effects of emphysema. The attempt to keep fundamentals in view throughout the book is commendable, but at times the thread is temporarily lost and the possession of a little specialized knowledge rather taken for granted. The book contains rather more information about the management of chest cases than the title might suggest, and therefore should be available not only to nursing and junior medical staff and physiotherapists in a chest department, but also in physical medicine, anaesthetic, and diagnostic X-ray departments, and, of course, schools of physiotherapy.

D. R. L. NEWTON

OBITUARY

J. B. MENNELL, M.A., M.D.(Cantab.)

IT is with deep sorrow that at the moment of going to press we learn of the death of one of the founder members of the British Association of Physical Medicine.

Dr. Mennell, who died on March 2 at the age of 77, qualified from St. Thomas's Hospital in 1908, and gave his services there uninterruptedly until he retired on account of ill health in 1935. He held various posts before being appointed medical officer in charge of the Physiotherapy Department, but no matter what these were, he invariably devised some measure aimed at obtaining early functional recovery in his patients. For his pioneer work in what was rehabilitation in its embryonic form the American Congress of Physical Medicine awarded him their Golden Key, an insignia he proudly wore on his watch chain.

His friends and the generations of trainees in physical medicine and physiotherapy who are the richer for having had him as a teacher and guide will mourn this great man whose guiding principle was "movement is life".

Members of the B.A.P.M. will wish to extend their sympathy to Mrs. Mennell and her family in their irreparable loss.

P. BAUWENS

ABSTRACTS OF THE LITERATURE

The Effect of Body Heating on the Circulation in Skin and Muscle. O. G. EDHOLM, R. H. FOX, and R. K. MACPHERSON. *J. Physiol.*, 1956, **134**, 612.

It has long been known that central heating increases the blood flow in the limbs, but it has not been clear whether this increase takes place in the deep as well as the superficial tissues. In the experiments described in this paper the blood flow in both forearms was measured simultaneously with plethysmographs. Profound superficial vasoconstriction was induced in one forearm by adrenaline iontophoresis. On heating the trunk in a water bath it was found that the total forearm blood flow did not increase during the induced superficial vasoconstriction, even when the blood flow in the control arm was increased to four or five times the resting rate. It was then necessary to show that the ionization had not caused vasoconstriction of the deep vessels, and this was done by demonstrating a normal increase in blood flow after exercise. It is concluded that the response in the peripheral circulation is confined to the skin and superficial tissues.

D. A. BREWERTON

The Application of Progressive Resistance Exercise in Physiotherapy. I. J. MACQUEEN. *Physiotherapy*, 1956, **42**, 83.

The author emphasizes that to obtain maximal muscle hypertrophy and power the optimum combination of resistance, repetitions, and frequency of exercise for a given muscle or group of muscles must be carefully worked out. In 1951 DeLorme and Watkins advocated gradually increasing the resistance between each set of repetitions, noting the maximum weight that can be used just ten times (the ten repetition maximum or 10 R.M.) for any particular exercise. Experiments on rats both with normal and with partially denervated muscles have shown the superiority of progressive resistance exercise over the fractional and free systems.

The clinical applications of progressive resistance exercises are discussed, accompanied by excellent photographic illustrations.

L. D. BAILEY

Immobilization with Resin-impregnated Plaster Bandages. J. D. LOWELL. *J. Bone Jt Surg.*, 1956, **38A**, 427.

The use of resin-impregnated cast material, made up as a bandage of plaster-of-Paris, resin, and catalyst bonded to crinoline, was studied in 79 patients, representing 118 applications. The work was carried out in the orthopaedic department of the Children's Medical Center, Boston, a variety of conditions being treated, ranging from club-foot to scoliosis, for which jackets or spicas were made.

It was found that in making these appliances about half to two-thirds the normal amount of plaster-of-Paris was required, and that with their use nursing of the children was much easier. A great advantage was the increased permeability to X rays. On the other hand, when the casts were dry they were more difficult to split than ordinary plaster-of-Paris. Resin plaster cracked and laminated if dipped into warm water. Its setting time was slower than that of plain plaster-of-Paris; it was also more difficult to mould, and was resilient even after setting. Too firm rubbing delayed the setting time. If a cast was bivalved immediately, lamination and cracking occurred, but this tended to

heal. Resin plaster was also found to be very adhesive, tending to bind ordinary plaster-of-Paris. The total cost of the casts was about the same as that of ordinary plaster-of-Paris.

The authors conclude that resin plaster is most satisfactory for large casts and for casts which might become wet, such as club-foot casts and spicas for infants.

In the 79 cases studied no skin reactions occurred, but later one doctor and four patients developed skin rashes when in contact with the material.

EUAN DAWSON

Rheumatoid Arthritis and the Positive L.E. Cell Phenomenon. J. H. KIEVITS, J. GOSLINGS, H. R. E. SCHUIT, and W. HIJMANS. *Ann. rheum. Dis.*, 1956, **15**, 211.

L.E. cells were found in 17% of 488 cases of rheumatoid arthritis. This observation led to a statistical analysis of its clinical implications. The group with positive L.E.-cell phenomenon showed a higher frequency of splenomegaly, diseases of the lower respiratory tract, abnormal urinary sediment, anaemia, false positive tests for syphilis, and a high erythrocyte sedimentation rate which failed to fall after a first course of gold treatment. In individual cases of rheumatoid arthritis the positive L.E.-cell phenomenon was not related to a typical clinical picture.—*Authors' summary*

Rheumatoid Arthritis in a Population Sample. J. H. KELLGREN and J. S. LAWRENCE. *Ann. rheum. Dis.*, 1956, **15**, 1.

This is an interesting and important small-sample survey of 10% of an urban population, the total number surveyed being about 400, of the age group 50-59. The incidence of typical rheumatoid arthritis diagnosed on clinical criteria confirmed by X rays and/or differential sheep-cell agglutination test (D.A.T.) was 1% for men and 3% for women. When, however, mild and atypical cases were included the rate rose to 11% for men and 27% for women. Although the incidence clinically determined was markedly greater in women, abnormal D.A.T. values and X-ray appearances were equally distributed among the sexes. Out of 283 individuals who were considered to be free from clinical rheumatoid arthritis, 20 showed some abnormality in radiographs or D.A.T. suggestive of the condition.

The most important lesson to be drawn from this survey is the need for standardization of criteria in examining samples of population, as without this they would become meaningless or even misleading.

G. D. KERSLEY

Kerato-conjunctivitis Sicca and Rheumatoid Arthritis. M. THOMPSON and S. EADIE. *Ann. rheum. Dis.*, 1956, **15**, 21.

Evidence of Sjögren's syndrome was found in 30 (14·3%) of 210 cases of rheumatoid arthritis. The clinical findings in 18 patients with severe or moderate kerato-conjunctivitis sicca are described and treatment by means of artificial tears, cortisone and hydrocortisone drops, and sealing of the lacrimal puncta is outlined.

G. D. KERSLEY

Nature of Anaemia in Rheumatoid Arthritis—II. Survival of Transfused Erythrocytes in Patients with Rheumatoid Arthritis. W. R. M. ALEXANDER, J. RICHMOND, L. M. H. ROY, and J. J. R. DUTHIE. *Ann. rheum. Dis.*, 1956, **15**, 12.

In a previous paper (*Ann. rheum. Dis.*, 1955, **14**, 63) the authors have discussed the metabolism of iron in rheumatoid arthritis. They now report a study of the fate of transfused red cells in this disease,

as determined by the Ashby differential agglutination technique. They found as follows: "(1) Erythrocytes from healthy donors are eliminated from the circulation with abnormal rapidity in patients suffering from rheumatoid arthritis. (2) Erythrocytes from rheumatoid donors survive longer in rheumatoid recipients than cells from healthy donors. (3) Consecutive transfusions from healthy donors do not as a rule influence the speed with which these cells are destroyed in patients with rheumatoid arthritis. (4) The administration of ACTH in doses sufficient to suppress the clinical signs of the disease does not prolong the survival of normal erythrocytes in rheumatoid recipients. (5) Haematological improvement after the administration of intravenous iron is not accompanied by increased survival of normal erythrocytes in rheumatoid recipients."

G. D. KERSLEY

Chloroquine and Rheumatoid Arthritis: A Short-term Controlled Trial. A. FREEDMAN. *Ann. rheum. Dis.*, 1956, **15**, 251.

To assess the effectiveness of chloroquine in the treatment of rheumatoid arthritis 66 patients were divided at random into two groups; Group I (34 cases) were given 200–300 mg. of the drug daily, and Group II received dummy tablets and served as controls. The trial extended over a period of 16 weeks. There was a suggestion that the chloroquine-treated cases derived benefit, but the improvement was not statistically significant. Toxic symptoms were minimal.

G. D. KERSLEY

Acquired Resistance to Corticotrophins. H. F. WEST. *Ann. rheum. Dis.*, 1956, **15**, 124.

A group of 51 patients, all suffering initially from severe rheumatoid arthritis or ankylosing spondylitis, have been followed up during prolonged ACTH therapy, using various forms of highly purified intramuscular corticotrophin. The results were assessed clinically and by urinary corticosteroid assay. Of the 51 patients, 42 acquired resistance to one or more preparations, and 17 experienced an allergic reaction at the site of injection.

The author believes that most highly purified corticotrophins are allergenic and that this is due to the contaminating or "carrier" proteins they contain. Where a specially purified hormone, corticotrophin A_1, was used this was not observed. He emphasizes the urgent need for more highly purified preparations of ACTH.

G. D. KERSLEY

Progressive Fluctuating Muscular Rigidity and Spasm (Stiff-man Syndrome): Report of a Case and Some Observations in 13 Other Cases. F. P. MOERSCH and H. W. WOLTMAN. *Proc. Mayo Clin.*, 1956, **31**, 421.

The authors describe the first case of this syndrome seen by them in 1924 and proceed to analyse the findings in this and in 13 similar cases seen since. The features of the syndrome are the insidious onset of episodes of tightening of the muscles of the neck, the attacks gradually increasing in frequency, duration, and severity with the involvement of muscles becoming more widespread. Finally, the spastic stiffness becomes crippling, the patient falling as though made of wood.

The records of the 14 cases were similar. Family histories did not reveal anything significant. There was some variation in the rapidity of onset and in the degree and extent of muscular rigidity. Some patients complained of pain during the spasms. In four cases pain was of major importance. The age of onset was from 28 to 54 years. No common cause

was found on careful history-taking. With one exception the condition appeared to be progressive. Ten of the 14 cases had been misdiagnosed. The chief complaint was of stiffness in 11 cases and of spasms in three. The muscles first affected were usually, but not always, those of the trunk and neck. The involvement was usually symmetrical and always spread to other muscle groups. The distal muscles were little affected, and the facial muscles, including those of the jaw, escaped. There was difficulty in breathing in two cases, and in swallowing when lying down in one case. Neurological examination was essentially negative apart from the rigidity, which was variable. Bladder function was normal. In no case was a myotonic reaction observed. The patients were mentally normal. Full laboratory examination revealed little of note, except that in four cases reducing substances were present in the urine. Electromyographic findings were normal, as were those of muscle biopsy. Many different palliative treatments were tried, but none proved beneficial.

Whether the cause was to be found in the spinal cord or the basal ganglia or in abnormal metabolism the authors were unable to decide.

D. C. ARNOTT

Periodic Benign Synovitis. A. D. WEINER and R. K. GHORMLEY. *J. Bone Jt Surg.*, 1956, **38A**, 1039.

Under this title the authors review 47 cases (33 in females and 14 in males) of idiopathic intermittent hydrarthrosis seen between 1930 and 1954 at the Mayo Clinic. The knee-joints were involved most frequently, but in 4 cases the knees escaped altogether and other joints, such as hips, ankles, hands, and feet, were affected. Thirty-seven patients were observed for a period of 10 to 47 years following the onset of their condition; over half these patients were free from symptoms when last seen. No patients in this series developed rheumatoid arthritis. Nine patients experienced remission during pregnancy, but attacks were not directly related to the menses or relieved at the menopause.

Laboratory investigations revealed normal white cell counts, normal haemoglobin levels, and only occasional elevation of the erythrocyte sedimentation rate. X-ray findings were negative. Synovial membrane obtained from the knees of 10 patients by needle biopsy or surgical incision was usually hyperaemic and oedematous with villous formation. Microscopically, the synovial membrane was often, but not always, infiltrated with plasma cells and lymphocytes. The microscopic picture in all cases was compatible with, but not necessarily diagnostic of, rheumatoid arthritis.

Many treatments were employed, including steroid therapy. These were mostly unsuccessful, although a few patients benefited from synovectomy and one patient attributed his remission to radiotherapy.

The authors conclude that: (1) these patients may have a rare form of rheumatoid arthritis; (2) the prognosis is much better than has previously been thought, as complete freedom from attacks without residual deformity can be expected in approximately half the cases; (3) excision of the synovial membrane may benefit those with severe involvement of the knee-joint, though the condition may recur; and (4) radiotherapy merits a further trial.

[All interested in this condition are recommended to read this paper in its entirety.]

S. MATTINGLY